

Seminar title MA461- Influence of sex and female hormones on performance and response to training

Reference: M M Samson 1, I B Meeuwsen, A Crowe, J A Dessens, S A Duursma, H J Verhaar (2000). Relationships between physical performance measures, age, height and body weight in healthy adults. *Age Ageing*. May;29(3):235-42.

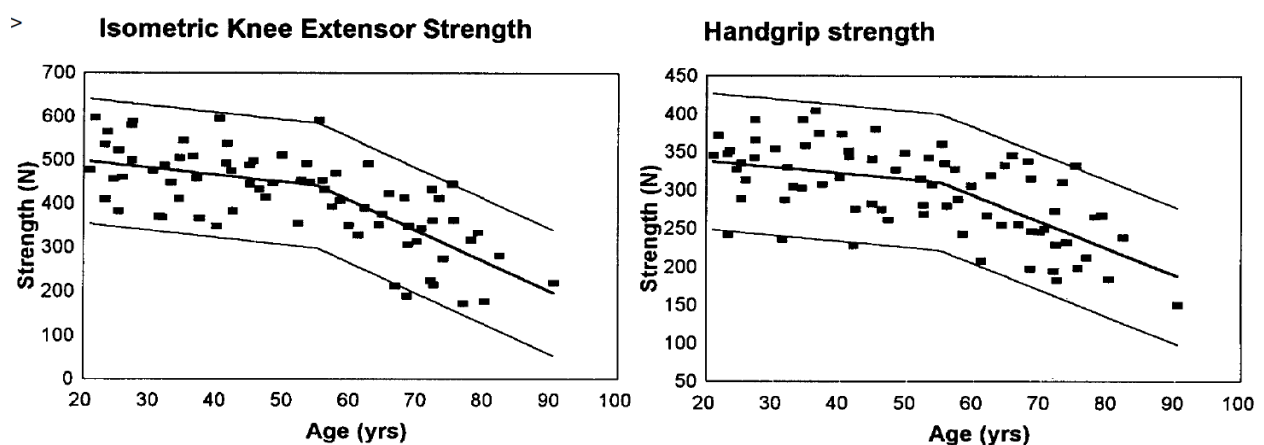


Figure : 1. Line of best fit with 95% confidence limits for the isometric knee extensor strength and handgrip strength among 74 healthy women aged between 20 and 90 years.

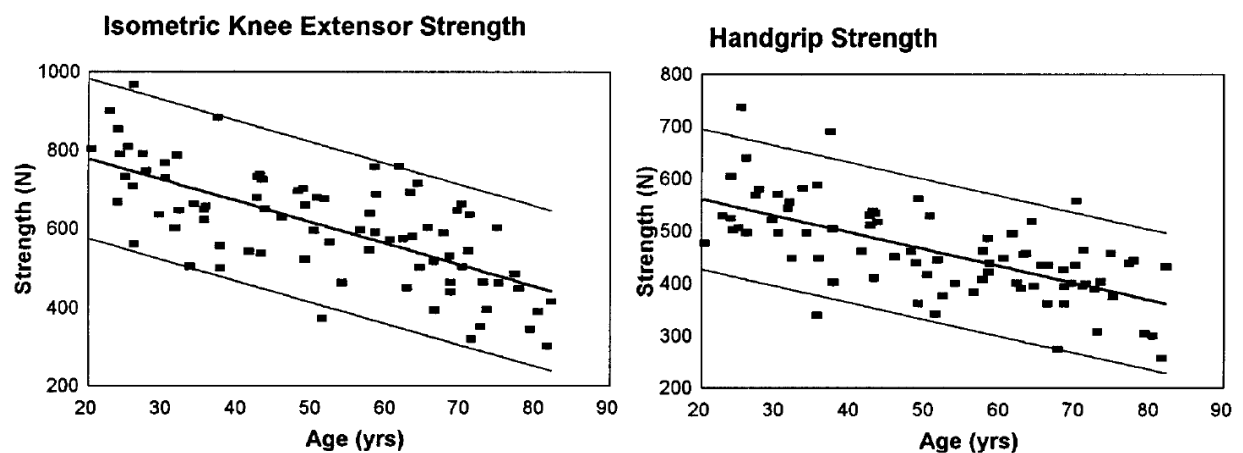


Figure 2. Line of best fit with 95% confidence limits for the isometric knee extensor strength and handgrip strength among 81 healthy men aged between 20 and 90 years.

Context: The figures are based on data from a cross-sectional study. The aim was to measure strength and functional mobility in healthy men and women over the adult age range to investigate the changes with age and sex. They were particularly interested in whether the menopause had any influence on muscle strength

Questions: Based on the results shown in the figures, what will you conclude in relation to their aim. Further, with reference to the curriculum for the present seminar, discuss potential mechanisms which can explain the observations in Samson et al (2000)

Answer guide:

Based on the figures from Samson et al. (2000), and the curriculum:

Conclusion from the figures:

The figures show isometric knee extensor and handgrip strength in men and women across different age ranges. Both strength parameters decline with age in men and women, but the decline is steeper in women, especially after the age of 50, likely around the menopause. This suggests that menopause may exacerbate muscle strength loss in women, as seen in the steeper decline in their muscle performance measures post-menopause compared to men.

Discussion of potential mechanisms:

The observed decline in muscle strength with aging, particularly in women post-menopause, can be explained by changes in hormonal levels, specifically the reduction in estrogen. According to the curriculum:

1. **Muscle Protein Metabolism:** Hansen (2018) highlighted that estrogen influences muscle protein metabolism, playing a protective role in maintaining muscle mass. With the reduction in estrogen during menopause, there is a decline in muscle protein synthesis, which may explain the accelerated strength loss.

2. **Satellite Cell Regulation:** Oxfeldt et al. (2022) discussed that sex hormones, including estrogen, regulate satellite cell activity, which is crucial for muscle repair and growth. The decrease in estrogen could impair satellite cell function, further contributing to muscle weakness and atrophy in postmenopausal women.
3. **Tendon and Connective Tissue:** Hansen et al. (2009) examined how estrogen affects tendon collagen synthesis and biomechanical properties. Estrogen decline could lead to weaker tendons and reduced force transmission efficiency, exacerbating the strength loss seen in aging women compared to men.

In summary, the menopause-related reduction in estrogen may lead to declines in muscle protein synthesis, impaired satellite cell function, and weakened tendon properties, all contributing to the observed steeper decline in muscle strength in women post-menopause, as seen in the figures from Samson et al. (2000).

Curriculum references used:

Oxfeldt M, Dalgaard LB, Farup J, Hansen M. "Sex Hormones and Satellite Cell Regulation in Women". Translational Sports Medicine. 14. April:2022:9065923

Hansen M, "Female hormones: do they influence muscle and tendon protein metabolism?", Proc Nutr Soc. 2018, vol 77, nr 1 (02), s 32-41

Hansen M, Kongsgaard M, Holm L, Skovgaard D, Magnusson SP, Qvortrup K, Larsen JO, Dahl M, Serup A, Frydystyk J, Flyvbjerg A, Langberg H, Kjaer M, "Effect of estrogen on tendon collagen synthesis, tendon structural characteristics, and biomechanical properties in postmenopausal women", J Appl Physiol, 2009 Apr;106(4):1385-93

Muscular and functional adaptations to low-load blood flow resisted exercise

Reference: Davids, C. J., Næss, T. C., Moen, M., Cumming, K. T., Horwath, O., Psilander, N., Ekblom, B., Coombes, J. S., Peake, J., Raastad, T., & Roberts, L. A. (2021). Acute cellular and molecular responses and chronic adaptations to low-load blood flow restriction and high-load resistance exercise in trained individuals. *Journal of Applied Physiology*, 131(6), 1731-1749.

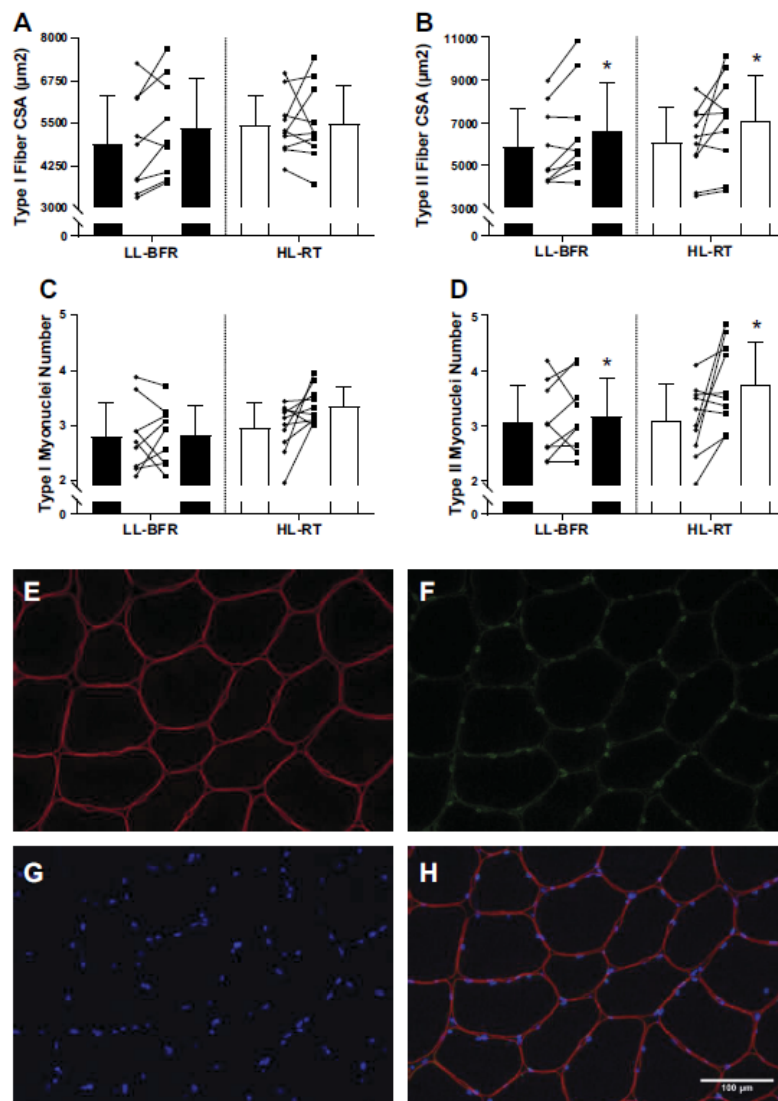


Figure 2. Changes in type I (A) and type II (B) fiber areas and myonuclear number per type I (C) and type II (D) fibers. Representative immunofluorescence images of muscle fiber membranes stained red for dystrophin (E) and myonuclei stained green for PCMI (F) and blue for DAPI (G). Nuclei within skeletal muscle were classified as myonuclei if they were positive for PCMI and DAPI, and the geometrical center was located within the dystrophin ring (H). Scale bar = 100 μm . Error bars represent standard deviation. *Significant change from baseline ($P < 0.05$). CSA, cross-sectional area; HL-RT, high-load resistance training ($n = 10$); LL-BFR, low-load resistance exercise with blood flow restriction ($n = 9$).

Context: The figure is from the study of Davids et al. 2021, with results from analyses on muscle biopsies obtained before and after the training intervention described briefly below.

Twenty-one males and females (means $\pm$ SD: 24.3 ± 3.1 yr) experienced with resistance training (4.9 ± 2.6 yr) performed 9 wk of resistance training (three times per week) with either High-Loads (75%–80% 1RM; HL-RT), or Low-Loads with Blood Flow Restriction (30%–40% 1RM; LL-BFR). Before and after the training intervention, resting muscle biopsies were

collected, and quadriceps cross-sectional area (CSA), muscular strength, and power were measured.

Question: Explain the theoretical relation between changes in myonuclear number and fibre cross-sectional area. Discuss the results shown in the figure against the differences in morphological and physiological effects normally shown for low-load blood flow restricted resistance exercise vs. traditional heavy-load resistance exercise!

Answer guide:

Theoretical relation:

Should be a short discussion on myonuclear domain and how the increase in myonuclei seems to be a prerequisite for substantial hypertrophy with strength training

Differences between LL-BFR vs. HL-RT:

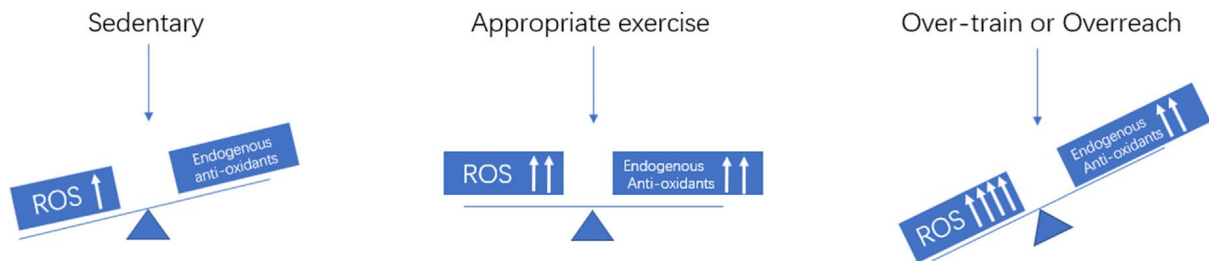
In most studies, LL-BFR seems to affect type I fibers more than type II fibers, whereas HL-RT has the opposite effect (type II fibers increase the most in size and myonuclear number). Consequently, it is surprising that LL-BFR did not result in increases in type I fiber area and myonuclear number, whereas type II fiber area increased. Importantly, the large variation in the individual responses and consequently the low statistical power should also be discussed, and a type II error may have masked an increase in type I fiber area in LL-BFR. Another surprise was the increase in myonuclear number in type I fibers with HL-RT, but this is not marked in the figure.

The load in the LL-BFR group was a bit higher than in previous studies, and it actually increased to 50% of 1RM at the end of the training intervention, and this might have contributed to a more marked effect in type II fibers with the LL-BFR protocol used in this study.

Other differences in morphological and physiological responses like more pronounced increases in capillaries and mitochondrial enzyme and thereby muscular endurance with LL-BFR can also be discussed.

The effect of nutritional interventions on muscle hypertrophy with strength training

Reference: Li, S., Fasipe, B., & Laher, I. (2022). Potential harms of supplementation with high doses of antioxidants in athletes. *Journal of Exercise Science & Fitness*, 20(4), 269-275.



Context: The figure shows a cropped version of a figure in Li et al. 2022. Three conditions are represented by a balance of ROS and endogenous antioxidants (ROS = reactive oxygen species).

Question: Explain the terms ROS and endogenous antioxidants (provide examples). Then, discuss why the balance may be as indicated under the three conditions.

Answer guide:

Terms:

ROS: Reactive Oxygen Species. These are highly reactive molecules that “steal” electrons from nearby molecules, thereby oxidizing them. Examples of ROS: superoxide (O_2^-) and hydrogen peroxide (H_2O_2). Production sites: ROS are produced at several locations in muscle fiber, e.g., in/near the sarcolemma, and in the mitochondria. Examples of enzymes that produce ROS are NADPH Oxidases (NOX family) and Xanthine Oxidase (XO). In the mitochondria, ROS are made in the electron transport chain (e.g., COX I, II).

Endogenous antioxidants are molecules that neutralize ROS by donating electrons. Key endogenous antioxidants include superoxide dismutase (SOD), catalase, and glutathione peroxidase. “Endogenous” means that the molecules are made in our cells, based on specific genes.

Endogenous antioxidants are different from exogenous antioxidants, which are not mentioned in the figure, but these, e.g., vitamin C and E, also have important functions in the ROS defence.

The balance:

ROS oxidize molecules. This can change the properties of the molecules and/or damage them. Under control of antioxidants, ROS may work as signaling molecules. During and in the hours after an endurance exercise session, PGC1alpha may be expressed and activated. Similarly, mTOR and/or MAPK might be activated during a strength training session. These are signaling crucial for adaptation to training, i.e., PGC1alpha induces mitochondrial biogenesis and mTOR/MAPK induces hypertrophy in skeletal muscles. Moreover, ROS also induce increased expression of endogenous antioxidants (via

NFkB, Nrf2, and/or AP-1), so that the ROS production is balanced by a sufficient antioxidant defense in healthy cells.

ROS can also damage molecules by oxidation. Damage molecules lead to inflammation and sometimes disease. Sedentary individuals may have too low levels of endogenous antioxidants compared to the ROS production. Similarly, athletes that train too much have overwhelming levels of ROS, even if their endogenous antioxidants levels are high. In both cases the balance is disturbed. Importantly, while short transient episodes of increased ROS is

Seminar title: The application of sport science knowledge in elite sport practice

Reference: Fullagar HHK, McCall A, Impellizzeri FM, Favero T, Coutts AJ. The Translation of Sport Science Research to the Field: A Current Opinion and Overview on the Perceptions of Practitioners, Researchers and Coaches. *Sports Med.* 2019 Dec;49(12):1817-1824.

“Research Areas of Interest to Coaches

When translating findings to practice, one needs to consider that coaches’ preferences are likely to be context specific, which can incorporate factors such as their background,

sport and level of competition. Thus, we propose an increased focus towards clearer definitions of sport science—how it is perceived and defined between different groups of stakeholders (coaches, players, researchers and practitioners). Interestingly, < 1% of surveyed coaches reported ‘sport science’ knowledge as necessary to be a better coach [25]. Indeed, there may be changes in what questions coaches want answered in sport science. For instance, if a coach has experience collaborating with researchers, they will likely yearn for different information compared with a coach who has little background or knowledge of what sport science research can offer. Ultimately, the coach is responsible for altering a positive behaviour change in an athlete and performance outcomes. Thus, practitioners and researchers should adopt methods and educate coaches on research areas that can elicit positive behaviour change to improve performance.”

Context: This is part of the article by Fullagar (2019) stating how coaches see the need for science support to become a better coach. They conclude that many coaches do not see the support of sport scientists to help them with their coaching.

To build such collaborations as fruitful for both the sport practitioners and athletes and the academia the gap between science and practice needs to be bridged.

Questions:

Explain why coaches see no need for sport science support to improve their coaching and assess the challenges and opportunities in translating sport science research into practical applications for coaches and athletes. What strategies can bridge the gap between researchers and practitioners to enhance the integration of scientific findings into sports practice?

Answer:

Coaches with limited knowledge on sport science typically do not know the possibilities scientists have to help them with their coaching. Sport science on the other side does often not know well enough the coaches real challenges which could guide them in conducting research that could help coaches with their work.

Challenges:

1. Communication Barriers:

- One of the primary challenges is the communication gap between researchers and practitioners. Researchers often use technical jargon and complex methodologies that may not be easily understood by coaches and athletes.

2. Relevance of Research:

- There is often a mismatch between the research conducted in laboratories and the practical needs of the field. Coaches and athletes require immediately applicable insights, whereas research can sometimes focus on niche or long-term issues.

3. Time Constraints:

- The fast-paced nature of sports seasons means that coaches and athletes may not have the time to wait for long research cycles. They require timely information to make decisions that impact performance immediately.

4. Resource Limitations:

- Implementing scientific findings often requires resources such as specialized equipment or additional staff training, which may not be available to all teams or athletes.

Opportunities:

1. Collaborative Research:

- Engaging coaches and athletes in the research process can ensure that studies are designed with practical applications in mind, increasing their relevance and applicability.

2. Technological Advancements:

- The use of technology in data collection and analysis can streamline the process of applying research findings, making it easier for practitioners to implement changes in training and competition.

3. Education and Training:

- Increasing the sport science literacy of coaches and athletes through workshops, seminars, and courses can empower them to better understand and integrate research findings.

4. Feedback Loops:

- Establishing systems for feedback from practitioners to researchers can help refine studies to better meet the needs of the field, creating a dynamic and responsive research environment.

Strategies to Bridge the Gap:

1. Interdisciplinary Teams:

- Forming interdisciplinary teams that include researchers, coaches, and athletes can facilitate the exchange of knowledge and ensure that research is both scientifically robust and practically applicable.

2. Knowledge Translation Frameworks:

- Implementing frameworks that focus on knowledge translation can help in systematically moving research findings from the lab to the field. This can include creating easily digestible summaries of research findings and practical implementation guides.

3. Regular Communication Channels:

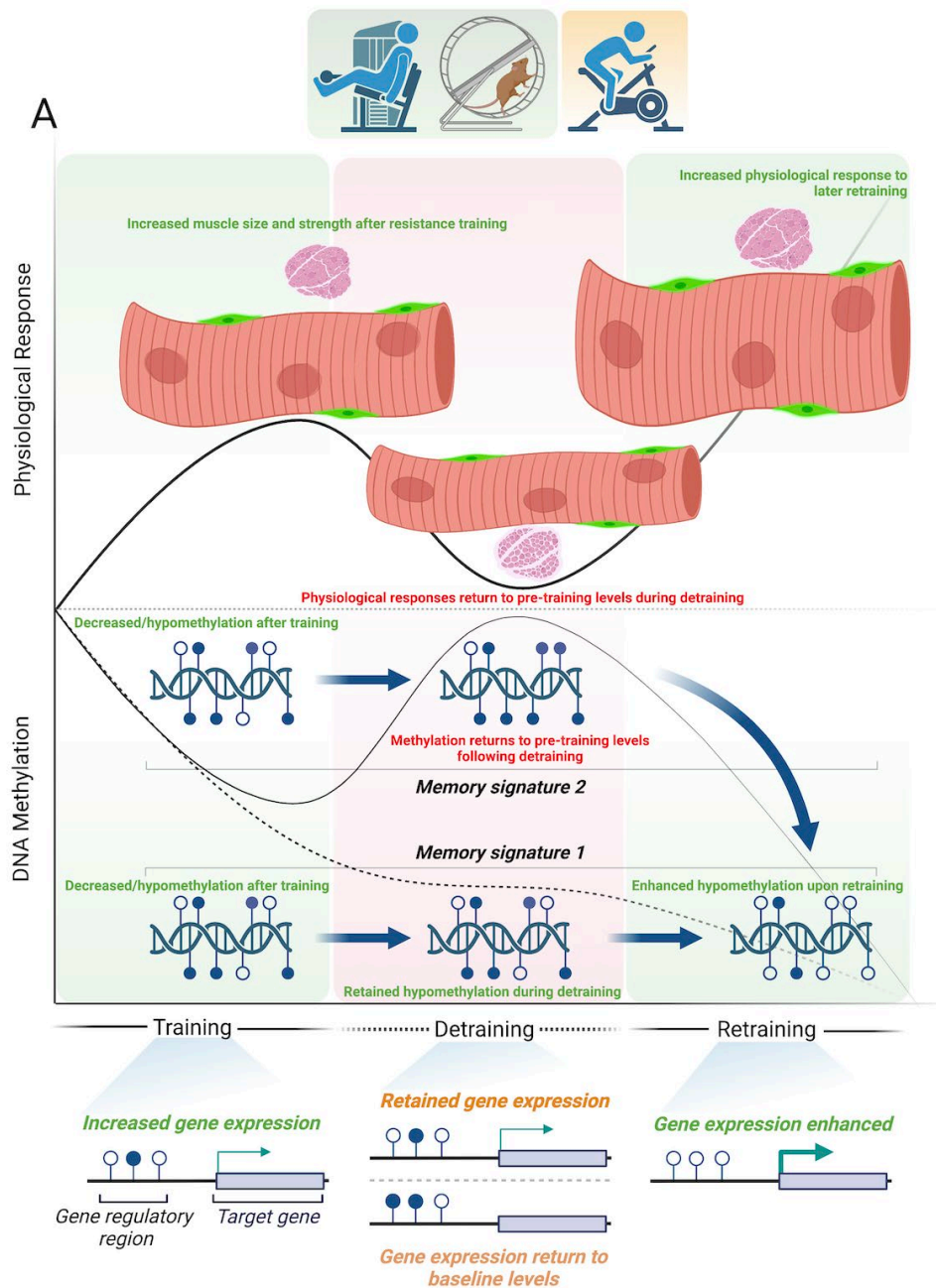
- Establishing regular meetings or communication platforms (such as webinars or online forums) can help maintain an ongoing dialogue between researchers and practitioners, ensuring that insights are shared in a timely manner.

In conclusion, while there are significant challenges in translating sport science research into practice, there are also numerous opportunities and strategies to overcome these barriers. By fostering collaboration, enhancing communication, and focusing on practical applicability, the

gap between researchers and practitioners can be effectively bridged, enhancing the integration of scientific findings into sports practice.

Does Skeletal Muscle have a Memory?

Reference: Sharples, A. P., & Turner, D. C. (2023). Skeletal muscle memory. *American journal of physiology. Cell physiology*, 324(6), C1274–C1294. <https://doi.org/10.1152/ajpcell.00099.2023>



Context: The above figure depicts the theory of a ‘positive’ epigenetic skeletal muscle memory following an earlier period of exercise training, followed by detraining and then after a later period of retraining.

Question: Describe, explain, and discuss positive epigenetic skeletal muscle memory.

In your answer, using the above figure as a guide, you should briefly state the main epigenetic modification associated with a positive epigenetic muscle memory studied to date (with a particular focus on exercise) and describe how alterations in this epigenetic modification effects gene expression and how this is associated with muscle memory. You should then briefly describe and explain some of the studies investigating positive epigenetic muscle memory and discuss how the findings of these studies support epigenetic memory.

Answer guide:

1. Describe epigenetic muscle memory and the main epigenetic modification associated with this- students could first define muscle memory in the context of ‘positive’ exercise adaptation. Can refer to ‘cell’ memory and retention of myonuclei in definition, but focus should be on epigenetic muscle memory and an explanation that retention of epigenetic modifications to muscle DNA after exercise training means that these genes expression levels are maintained during detraining and/or enhanced upon later retraining (these are the 2 predominant memory signatures in the figure provided). Higher level students may be able to describe how the mechanism of DNA methylation biochemically influences expression (methyl binding to CpG sites affecting transcription factor binding /accessibility, may also include e.g., regulatory enzymes controlling hypo (TET’s) and hypermethylation (DNMT’s)) and that there is an inverse relationship with methylation and expression. Students should be able to describe that exercise training evokes predominantly hypomethylation of genes and increased gene expression and that this hypomethylation is retained into detraining and or enhanced upon retraining.
2. Describe and explain 2-3 (out of 5) possible studies investigating a positive epigenetic muscle memory:
 - i) Seaborne, R. A., Strauss, J., Cocks, M., Shepherd, S., O'Brien, T. D., van Someren, K. A., Bell, P. G., Murgatroyd, C., Morton, J. P., Stewart, C. E., & Sharples, A. P. (2018). Human Skeletal Muscle Possesses an Epigenetic Memory of Hypertrophy. *Scientific reports*, 8(1), 1898. <https://doi.org/10.1038/s41598-018-20287-3> As the first study to demonstrate epigenetic memory in human skeletal muscle following repeated resistance training in young adults, demonstrating the 2 predominant epigenetic memory signatures described in points 2 and the figure provided.

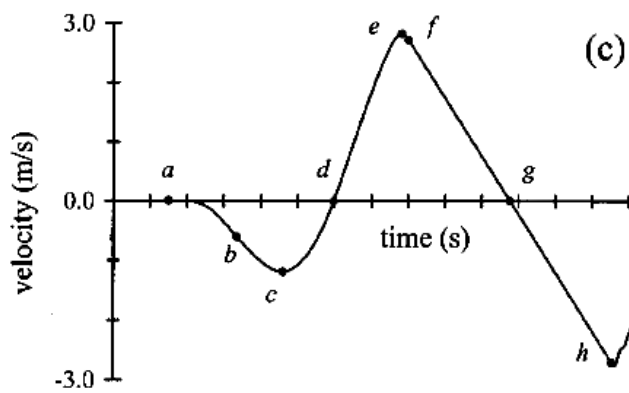
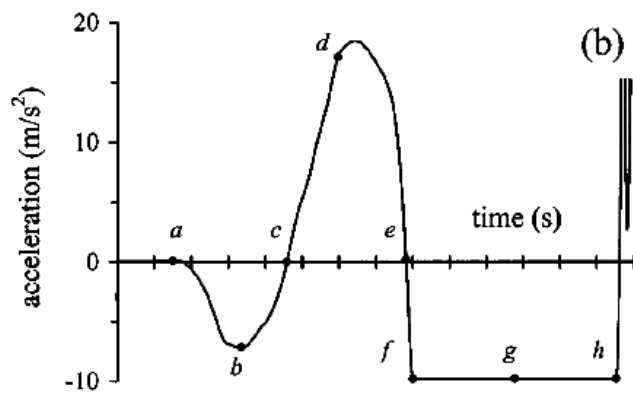
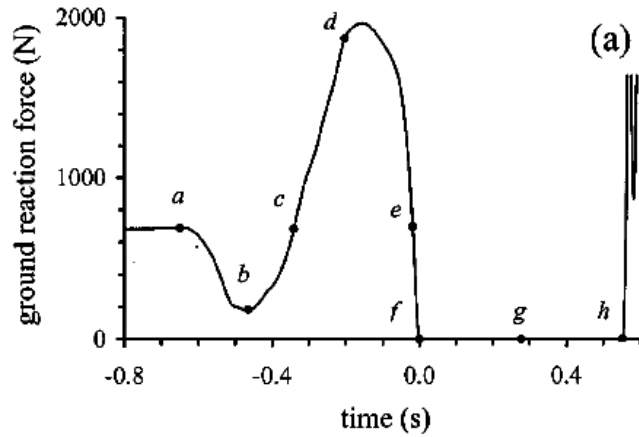
Can also include relevant/related study to i) above:

- ii) Turner, D. C., Seaborne, R. A., & Sharples, A. P. (2019). Comparative Transcriptome and Methylome Analysis in Human Skeletal Muscle Anabolism, Hypertrophy and Epigenetic Memory. *Scientific reports*, 9(1), 4251. <https://doi.org/10.1038/s41598-019-40787-0>
 - iii) Blocquiaux, S., Ramaekers, M., Van Thienen, R., Nielens, H., Delecluse, C., De Bock, K., and Thomis, M. (2022) Recurrent training rejuvenates and enhances transcriptome and methylome responses in young and older human muscle. *JCSM Rapid Communications*, 5: 10–32. <https://doi.org/10.1002/rco2.52>. Demonstrating epigenetic memory of repeated resistance training in elderly skeletal muscle.
 - iv) Wen, Y., Dungan, C. M., Mobley, C. B., Valentino, T., von Walden, F., & Murach, K. A. (2021). Nucleus Type-Specific DNA Methylomics Reveals Epigenetic "Memory" of Prior Adaptation in Skeletal Muscle. *Function (Oxford, England)*, 2(5), zqab038. <https://doi.org/10.1093/function/zqab038> that demonstrated epigenetic muscle memory after power weighted wheel running training, detraining in mice.
 - v) Pilotto, A. M., Turner, D. C., Mazzolari, R., Crea, E., Brocca, L., Pellegrino, M. A., Miotti, D., Bottinelli, R., Sharples, A. P., & Porcelli, S. (2025). Human skeletal muscle possesses an epigenetic memory of high-intensity interval training. *American journal of physiology. Cell physiology*, 328(1), C258–C272. <https://doi.org/10.1152/ajpcell.00423.2024>. Demonstrating epigenetic muscle memory after repeated HIIT training in adult human muscle.
- 3) For discussion of epigenetic memory studies above- could discuss the gene/gene pathways that demonstrate epigenetic memory in skeletal muscle and how this relates to muscle adaptation e.g., such as UBR5. OR discuss the role of cell/nuclei specific epigenetic modifications in muscle memory studied in Wen et al., that then may provide a synergistic memory with ‘cell’ memory/myonuclear retention (Reviewed in Sharples and Turner, 2023). OR provide a comparison of epigenetically modified gene pathways associated with divergent exercise/contraction stimuli such as resistance training vs. HIIT training that relates to alternate adaptation to these modes of exercise. Alternative discussion could also be on limitations/future directions of muscle memory research such as the role of latest technology (e.g. single cell/nuclei omics); aging and disease and retention of epigenetic modifications in muscle that can be reversed by exercise training or discussion of a potential ‘negative’ epigenetic memory e.g., periods of muscle loss/wasting are remembered and if encountered again makes muscle more protective or susceptible when a repeated encounter occurs, and suggested consequences for therapies counteracting muscle wasting. Also, future directions/applied implications around optimising or boosting a positive memory using different exercise intensities/modes to give muscle a longer lasting memory and/or optimising recovery in athletes could be included and finally perhaps relation of epigenetic tests to detect steroids even prior doping when individuals

are clean. If students discuss one or two of these points, then these **discussion** points and the detail contained in the answer could differentiate between a B or an A. Without a single discussion point listed in 3, students may be limited to a C or a B depending on how comprehensively they answered points 1 or 2 above.

Jumping capacity in testing and training

Reference: Linthorne, N. P. (2001). Analysis of standing vertical jump using a force platform. American Journal of Physics, 69(11), 1198-1204. <https://doi.org/10.1119/1.1397460>



Context: The figure shows the force-trace of a vertical jump. The ground reaction force (a) is converted to acceleration (b), and velocity (c).

Questions:

1. Explain what kind of jump is this?
2. Explain how acceleration (m/s^2) and velocity (m/s) can be derived from force (N).
3. Name the different positions or phases of the jump (a-h). “a” is the start of the jump.

Answer guide:

1. This is a CMJ. This can be seen from the negative acceleration of the center of mass that is followed by an immediate push-off phase.
2.
 - a. Newton's second law: $F = ma \Rightarrow a = F/m$.
 - b. Integrating acceleration over time gives velocity: $v(t) = \int a \, dt$
3. Phases/points:
 - a. a = start of the jump, b = maximal downward acceleration of the jumper's COM, c = the ground reaction force is equal to body weight, d = lowest point of the countermovement where the COM is momentarily at rest, e = the ground reaction force is again equal to body weight and the point of maximal upward velocity of the COM, f = take-off (0 N), g = the point where the COM is at maximal height and momentarily at rest, h = landing.

The physiology of high-intensity exercise

References (2 articles):

Gaitanos, G. C., Williams, C., Boobis, L. H., & Brooks, S. (1993). Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol* (1985), 75(2), 712-719.

Parolin, M. L., Chesley, A., Matsos, M. P., Spriet, L. L., Jones, N. L., & Heigenhauser, G. J. (1999). Regulation of skeletal muscle glycogen phosphorylase and PDH during maximal intermittent exercise. *American Journal of Physiology*, 277(5), E890-900.

Article excerpts:

Gaitanos et al (1993):

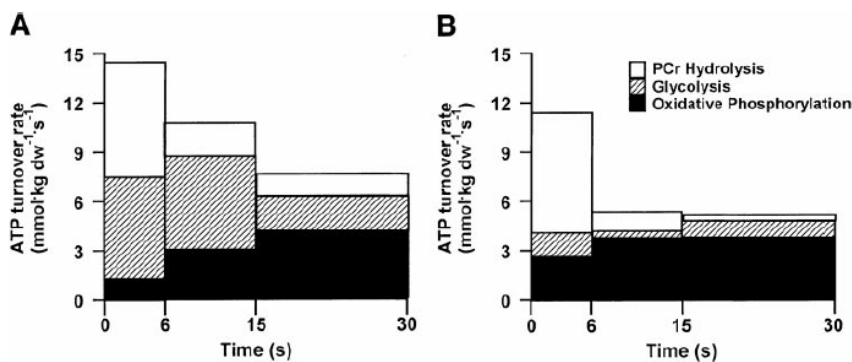
TABLE 2. Estimates of ATP production from anaerobic sources during first and final 6-s sprint of exercise test

	ATP Production, mmol/kg dry wt		ATP Production Rate, mmol · kg dry wt ⁻¹ · s ⁻¹	
	<i>Sprint 1</i> (n = 8)	<i>Sprint 10</i> (n = 7)	<i>Sprint 1</i> (n = 8)	<i>Sprint 10</i> (n = 7)
Total	89.3±13.4	31.6±14.7	14.9±2.2	5.3±2.5
From glycolysis	39.4±9.5	5.1±8.9	6.6±1.6	0.9±1.5
From PCr	44.3±4.7	25.3±9.7*	7.4±0.8	4.2±1.6

Values are means ± SD; n, no. of subjects. * Significant difference from *sprint 1*, *P* < 0.01.

Parolin et al (1999):

Fig. 7. ATP turnover rates from PCr hydrolysis, glycolysis, and oxidative phosphorylation during each time interval in the first (A) and third (B) bouts of maximal isokinetic cycling.



Context: In Gaitanos et al (1993), participants carried out 10 6-second sprints with 30-second passive rest in between, and muscle biopsies were taken before and after the first and 10th sprint (“Table 2”). In Parolin et al (1999), three 30-second all-out exercises were carried out with 4-minute passive rest in between, and biopsies were taken before, at 6, 15 and 30 seconds during the first (“Figure 7A”) and 3rd bout (“Figure 7B”). In both studies, muscle metabolites were measured to calculate anaerobic ATP production/turnover rates by phosphocreatine (PCr) hydrolysis and by glycolysis. ATP turnover rate from oxidative phosphorylation was calculated in the study by Parolin et al. (1999) but not in the study by Gaitanos et al. (1993). Mean power output dropped by about 25% from the first to the final exercise bout in both studies, and you should assume a similar ATP demand per Watt on the cycle ergometer during all exercise bouts in both studies for simplicity.

Questions: Use the above data and description to answer the following:

1. Approximately, how large a fraction of the total anaerobic ATP turnover is coming from PCr hydrolysis and glycolysis during a single 6-seconds and a single 30-seconds all-out test? Why is this different between these two tests?
2. How is the balance between ATP turnover from PCr hydrolysis and glycolysis altered when several all-out tests are repeated interspersed by short recovery periods, and why?
3. During the first 6-seconds of all-out exercise in Parolin, PCr hydrolysis is unaltered from the 1st to the 3rd bout, whereas being reduced by 45% in Gaitanos et al. (1993) from the 1st to 10th 6-second sprint. Why is this reduction different between these two studies?
4. Why was the mean power output reduced by only 25% from sprint 1 to sprint 10 in Gaitanos et al. (1993) when the anaerobic ATP turnover was reduced by as much as 65%?

Answer guide:

1. Of the total anaerobic ATP production, PCr hydrolysis accounts for 50%, glycolysis for 44% and ATP hydrolysis for 6% during a 6-second sprint (*if something close to 50/50 is written here, this is approved. ATP hydrolysis does not need to be mentioned*). During 30-second all-out exercise, a larger fraction comes from glycolysis due to increased glycogenolysis and reduced PCr hydrolysis between 6-30 seconds of exercise. This is because of higher glycolytic flux/glycolytic enzyme activation and/or due to partial depletion of limited PCr stores (around 60%/40% comes from glycolysis/PCr hydrolysis).
2. The glycolytic ATP production (glycogen degraded to lactate) is reduced more than PCr hydrolysis, and causes the fractional contribution from PCr hydrolysis to be dramatically increased. This is mainly caused by two mechanisms: 1) Glycolysis is inhibited due to metabolite accumulation and

acidosis from prior exercise bouts and 2) a higher mitochondrial activation causes pyruvate to be oxidised (instead of reduced to lactate), which reduces the glycolytic anaerobic component. (Not needed to write to get full score but can substitute the prior: An alternative explanation, which is also valid, is that during high-intensity exercise, when PCr is available it is used as a priority source for ATP resynthesis, reducing the necessity of relying on glycolysis and lactate production. The fast recovery of PCr is critical for reducing the reliance on glycolysis as an energy source).

3. In Parolin et al. (1999), only three exercise bouts were performed with relatively long recovery periods (4 minutes). Whereas in Gaitanos et al (1993), the 6-second sprints are repeated 10 times with a short recovery in between (30 seconds). Therefore, the PCr stores are almost fully recovered and PCr levels are higher before the final exercise bout in Parolin, making the anaerobic energy yield from PCr hydrolysis higher.
4. The fact that power output was better maintained than the anaerobic energy yield can be explained by an increased contribution by oxidative phosphorylation (oxidative ATP production in the mitochondria) from the first to the final sprint.

Introduction to Musculoskeletal Modelling

Reference: Rice, H., Starbuck, C., Willer, J., Allen, S., Bramah, C., Jones, R., Herrington, L., & Folland, J. (2024). Does high-intensity running to fatigue influence lower limb injury risk? *Journal of Science and Medicine in Sport*, S1440-2440(24)00490-0.

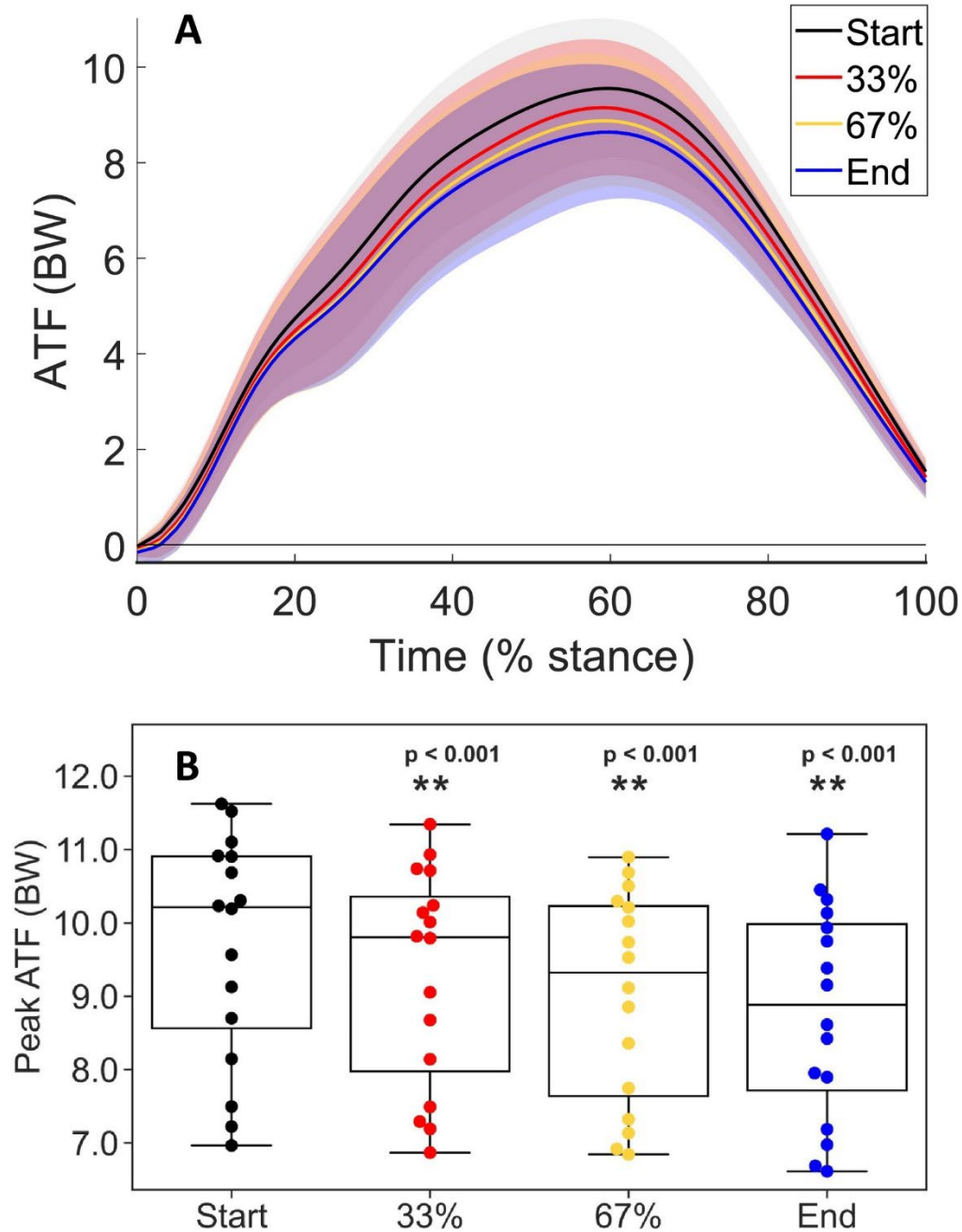


Fig. 1. A: Time-normalised Achilles tendon force (ATF) at four time points during the run. Time series lines represent the means; shading represents the standard deviations. B: Box plot displaying peak Achilles tendon force at each of the four time points during the run ($n = 16$). Box plots represent the median and 1st and 3rd quartiles whilst the error bars represent the range, excluding outliers (none present). Mean values for each participant are overlaid in filled circles as a swarm plot at each time point. **Significantly different ($p < 0.001$) from Start.

Context: The authors conducted a study to quantify the changes in loading of the Achilles tendon during an exhausting high-intensity run to fatigue at middle-distance speed. It was hypothesised that there would be no change in Achilles tendon loading as the run progressed. Achilles tendon force was estimated by dividing the sagittal ankle joint moment by the Achilles tendon moment arm. Achilles tendon moment arms were computed from ankle joint angles based on regression equations.

Questions: Do the results support the authors hypotheses? The last two sentences of the text above outline the model used to represent the Achilles tendon. How could the complexity of this model be increased, and what are the potential advantages and disadvantages of increasing the complexity?

Answer guide: The results are in contrast to the hypothesis as it was seen that peak Achilles tendon force decreased throughout the run. The model complexity could be increased by using participant-specific properties of the tendon, such as measured moment arms, and material properties of the tendon, and estimating Achilles tendon stress or strain. However, in a study design such as this one where within-individual changes are being quantified, it may not be beneficial to increase the complexity. The disadvantages of increasing the complexity are that it requires more participant-specific data which may be difficult or expensive to obtain, and much greater computation time may be required.

Seminar title: Training load monitoring in team sport

Reference: Hulin BT, Gabbett TJ, Blanch P, et al Spikes in acute workload are associated with increased injury risk in elite cricket fast bowlers. British Journal of Sports Medicine 2014;48:708-712.

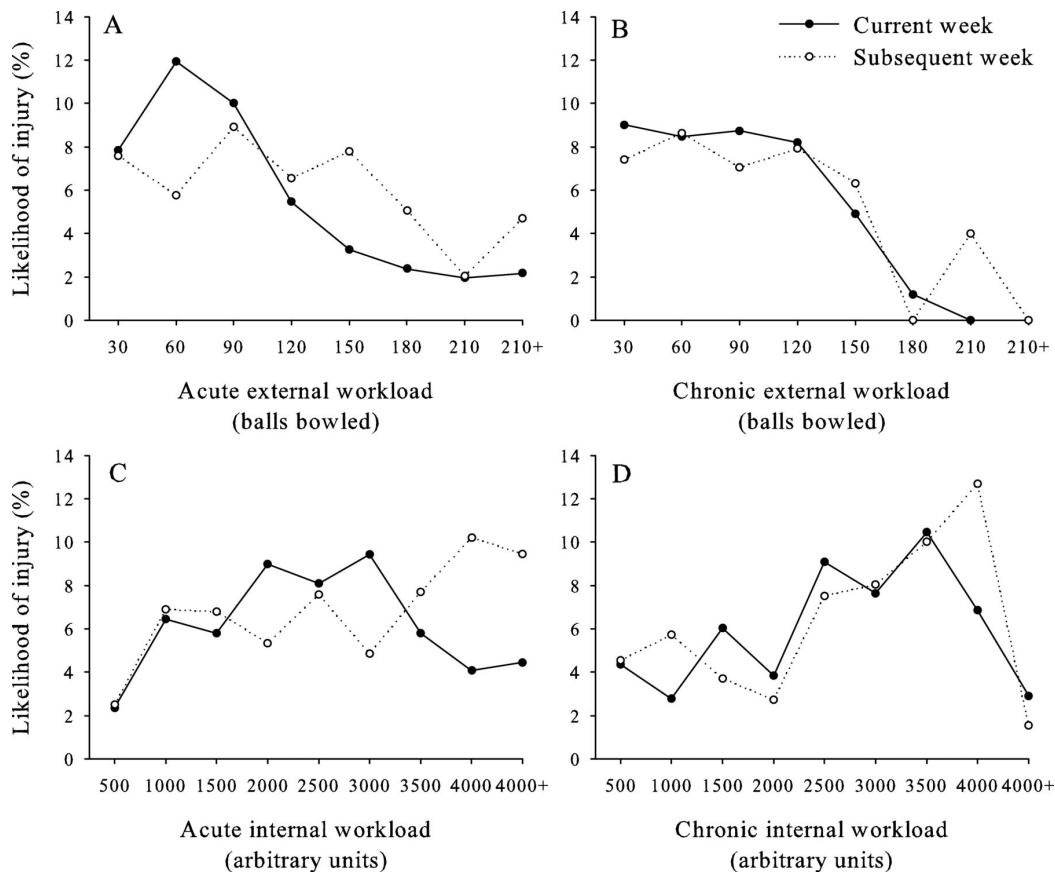


Figure 1: Likelihood of injury at acute (A) and chronic (B) external workloads, and acute (C) and chronic (D) internal workloads.

The relationships between injury risk and acute and chronic external workloads are shown in figure 1A,B, respectively. There was a relationship ($p=0.0001$) between acute external workloads in the current week and injury, with higher external workloads associated with a lower injury risk. No relationship ($p=0.172$) was found between acute external workloads and injury in the subsequent week. The likelihood of injury in response to acute and chronic internal workloads is shown in figure 1C,D, respectively. No relationships were found between either acute ($p=0.176$) or chronic ($p=0.210$) internal workloads and injury in the current week, or between acute ($p=0.109$) or chronic ($p=0.381$) internal workloads and injury in the subsequent week.

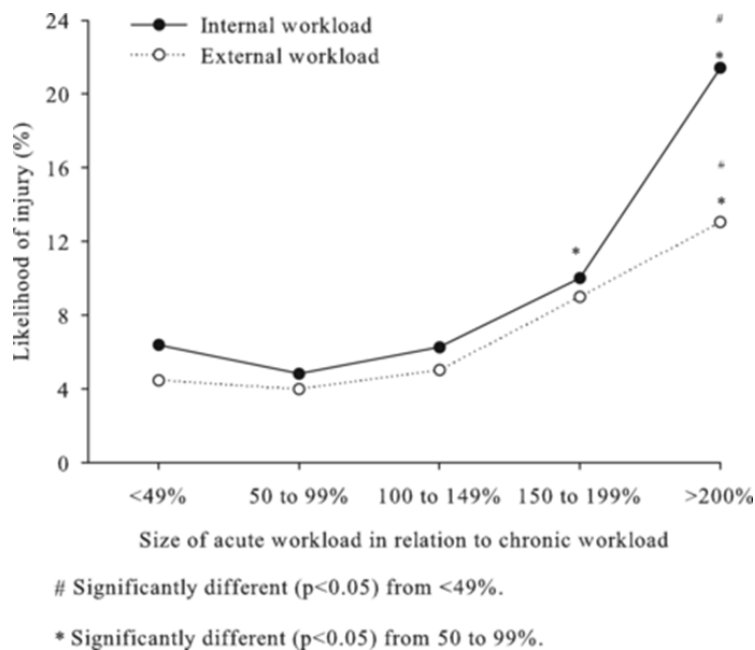


Figure 2: Likelihood of injury in the subsequent week for positive and negative training-stress balance ranges.

Context: The above figures were published in a study investigating the relationship between training load and injury risk. The authors conclude that a high chronic external workload is protective of injury, and that the monitoring and comparison of acute and chronic workloads can offer valuable insight into the likelihood of injury.

Question: Use the figures to explain the conclusions of the authors

Answer: The authors' conclusions are based on the relationships observed between training load (both acute and chronic) and injury risk. They show that a higher chronic external workload (Figure 1B) was protective against injury, supporting the idea that athletes with a greater long-term training load are better adapted and less susceptible to injuries. This aligns with the principle that well-conditioned athletes can handle greater workloads with lower injury risk.

Figure 2 illustrates the likelihood of injury (%) based on the size of acute workload in relation to chronic workload, comparing internal (solid line) and external (dotted line) workloads. The graph shows that with low to moderate Acute-Chronic workload (<49% - 149%) the injury risk is relatively low and stable for both internal and external workloads, however in high Acute-to-Chronic Workload Ratio (150–199%) the injury risk increases significantly (compared to 50–99%). In very high Acute-to-Chronic Workload Ratio (>200%) there is a sharp rise in injury likelihood. Significant differences from both the <49% and 50–99% groups indicate a correlation between workload spikes and injury risk. Based on this, comparing acute and chronic workloads can offer valuable insight

into the likelihood of injury – where a higher Acute-to-Chronic Workload Ratio is shown to have a higher injury risk compared to lower Acute-to-Chronic Workload Ratio.

Understanding forces in the context of running and human movement

Reference: Willer, J., Allen, S. J., Burden, R. J., & Folland, J. P. (2024). How Humans Run Faster: The Neuromechanical Contributions of Functional Muscle Groups to Running at Different Speeds. *Scandinavian Journal of Medicine & Science in Sports*, 34(8), e14690.

FIGURE 1

Positive work by flexion and extension moments of the ankle, knee, and hip during stance, swing and overall stride. Data are group mean $\pm$ SD ($n = 11$) and unilateral, average of both legs.

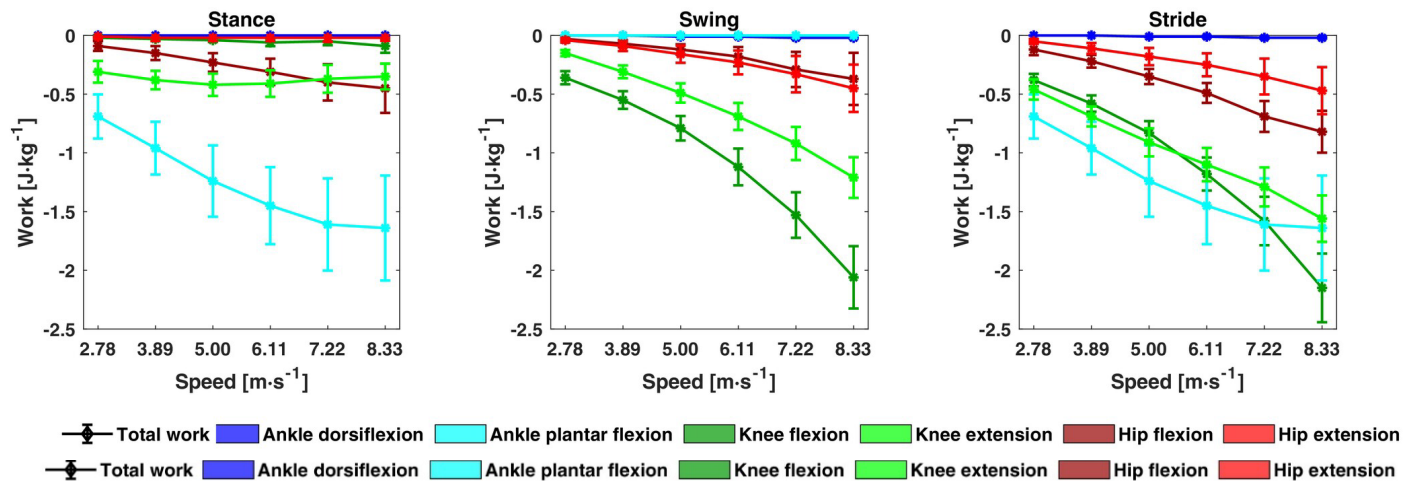


FIGURE 2

Negative work by flexion and extension moments of the ankle, knee, and hip during stance, swing and overall stride. Data are group mean $\pm$ SD ($n = 11$) and unilateral, average of both legs.

Context: The authors of this study investigated the sagittal plane joint contributions to increasing running speeds amongst middle-distance runners. It was hypothesized that during a whole stride cycle: (i) energy generation (positive work) at slow running speeds would be dominated by the ankle plantar flexors, but this would plateau with increasing speed, and be overtaken by the hip extensors as the largest energy generator at high speeds; (ii) the primary energy absorber (negative

work) would be the ankle plantar flexors at low speeds, but this would be exceeded by the knee flexors at high speeds.

Questions: Explain whether the data appear to support their hypotheses and summarize what was observed during only the stance phase (i.e. instead of the stride phase).

Answer guide: As hypothesized, Figure 1 demonstrates that the positive work increased in the plantar flexors and hip extensors but this leveled off in the plantar flexors at higher speed whereas it continued to increase in the hip flexors such that they became the greatest contributors. The 'Stride' figure in Figure 2 shows that the ankle plantar flexors contribute the greatest negative work at slow speeds but this is overtaken at the fastest speed by the knee flexors.

During stance, there was limited contribution to positive work from ankle dorsiflexion, knee flexion and hip flexion. The greatest contributor were the ankle plantar flexors. A similar trend was observed in terms of negative work, whereby the extensors (including ankle plantar flexors) were dominant during stance with the ankle contributing the greatest magnitude of work.

Seminar title: Stretching exercise. What is it good for?

Acute and long-term effects of stretching in relation to function, performance and injury risk

Reference: Anthony D Kay, Anthony J Blazeovich. Effect of acute static stretch on maximal muscle performance: a systematic review. Med Sci Sports Exerc. 2012 Jan;44(1):154-64.

Figure A:

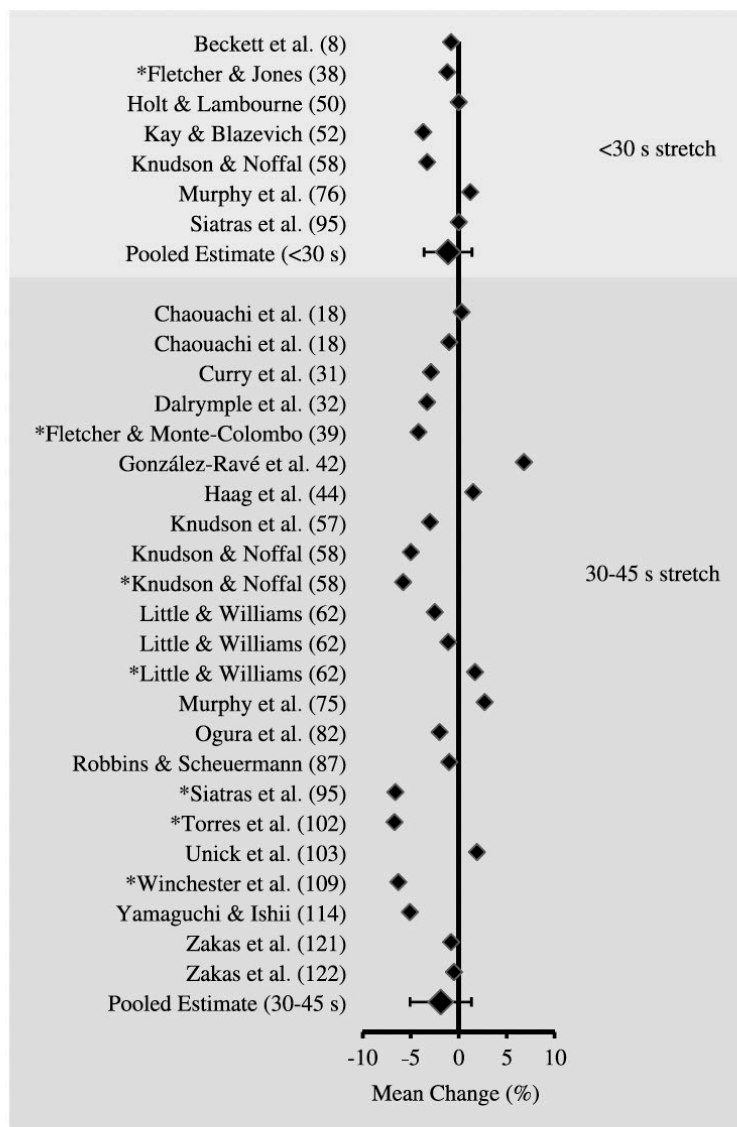
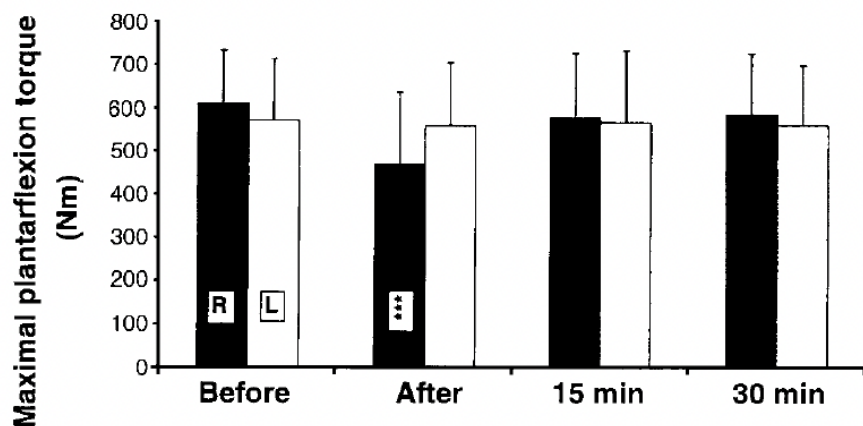


FIGURE 1—Mean percentage change (* $P < 0.05$, significant) in strength-, power-, and speed-dependent task performances after stretches of <30 s (*top*) or 30–45 s (*bottom*) in duration. Most studies found no significant reduction in muscle performance after shorter stretch durations, with small mean reductions calculated across studies indicating no meaningful change in performance.

Figure B:



Context: Despite that stretching is routinely applied as part of warm-up routines, it has in been questioned if stretching exercise should be performed prior to maximal performance efforts (e.g. competition). This discussion is based on a number of studies that demonstrated reduced muscle-contraction performance following stretching.

Figure B above, is one such study that measured leg strength (maximal plantarflexion torque) prior to, acutely, and 15 and 20 min after 1 hr of cyclic stretching performed unilaterally for the right (R) leg (L, left leg served as control) (J Avela, H Kyröläinen, P V Komi. Altered reflex sensitivity after repeated and prolonged passive muscle stretching. J Appl Physiol 1999 Apr;86(4):1283-91). More recently a review was conducted that examined the acute effect of stretching on strength, power and speed performance after stretching of less duration (Figure A above).

Question: Discuss briefly the effects of acute and habitual stretching. Explain the findings that are summarized in the two figures above (A & B) and based on this, give your recommendation to if and specifically how athletes should include stretching as part of their training/warm up routines prior to competition.

Answer: Habitual stretching (e.g. repeated stretching exercise conducted regularly (daily or 3-4/week) for 3-8 weeks +) is well known to increase joint range of motion (ROM) over time, an effect that is mainly attributed to stretch tolerance, and reversible if stretching exercise is terminated. Similarly, it is known that one stretching bout acutely increases range of motion (ROM), but also reduces maximal voluntary contraction strength, sprint speed, jump height. However, the acute reduction in contractile performance is mostly observed following longer stretches +1-5 min, are transitory and last only minutes following the stretch. As shown in figure B, 1 hr. of stretching reduced maximal plantarflexion torque approximately 20% (R, right leg) following the stretch, but 15 min later the maximal torque was restored. Figure A is a Forest plot, summarizing the acute effect of brief stretching efforts (duration <30 sec and 30-45 sec) on strength and power performance. It seems, from the single studies and from the pooled estimate that brief stretching has no meaningful negative effect on contractile performance. As a

coach or trainer, the recommendation would consequently be that stretching can be a part of the warm-up routine prior to performance, but excessive stretching (duration above 1 min) for the single joint/muscle should be avoided.

Seminar title: Musculoskeletal connective tissue: function and adaptation to training

Reference: Couppé, C., Kongsgaard, M., Aagaard, P., Hansen, P., Bojsen-Moller, J., Kjaer, M., & Magnusson, S. P. (2008). Habitual loading results in tendon hypertrophy and increased stiffness of the human patellar tendon. *Journal of Applied Physiology*, 105(3), 805–810.

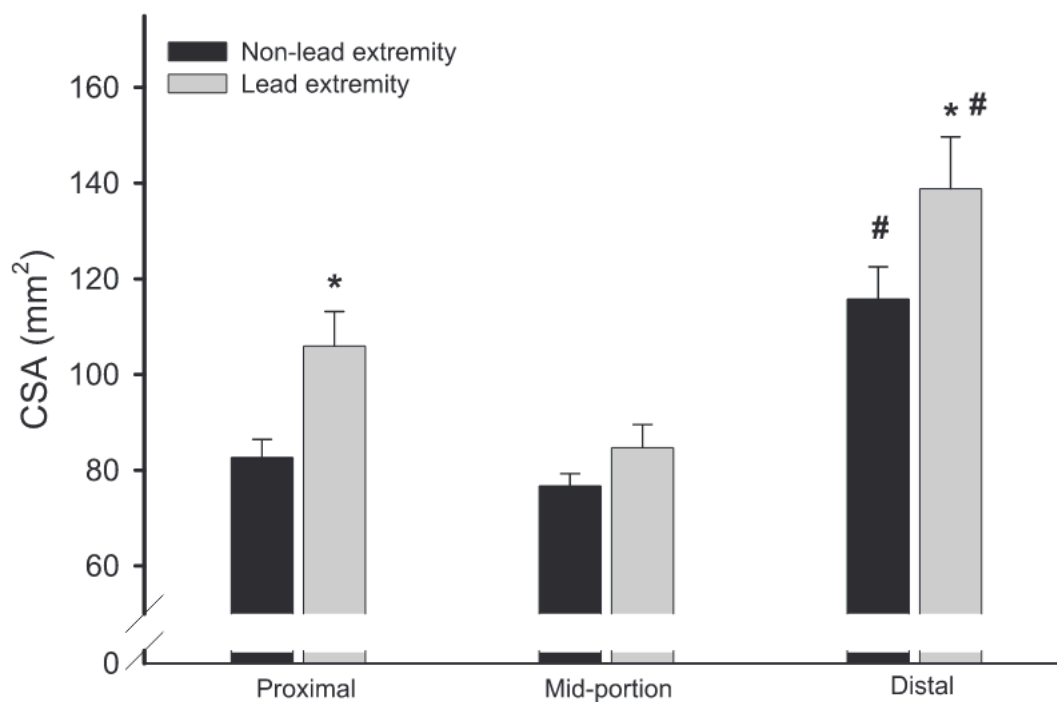


Fig. 2. Patellar tendon CSA distribution. Values are means $\pm$ SE.

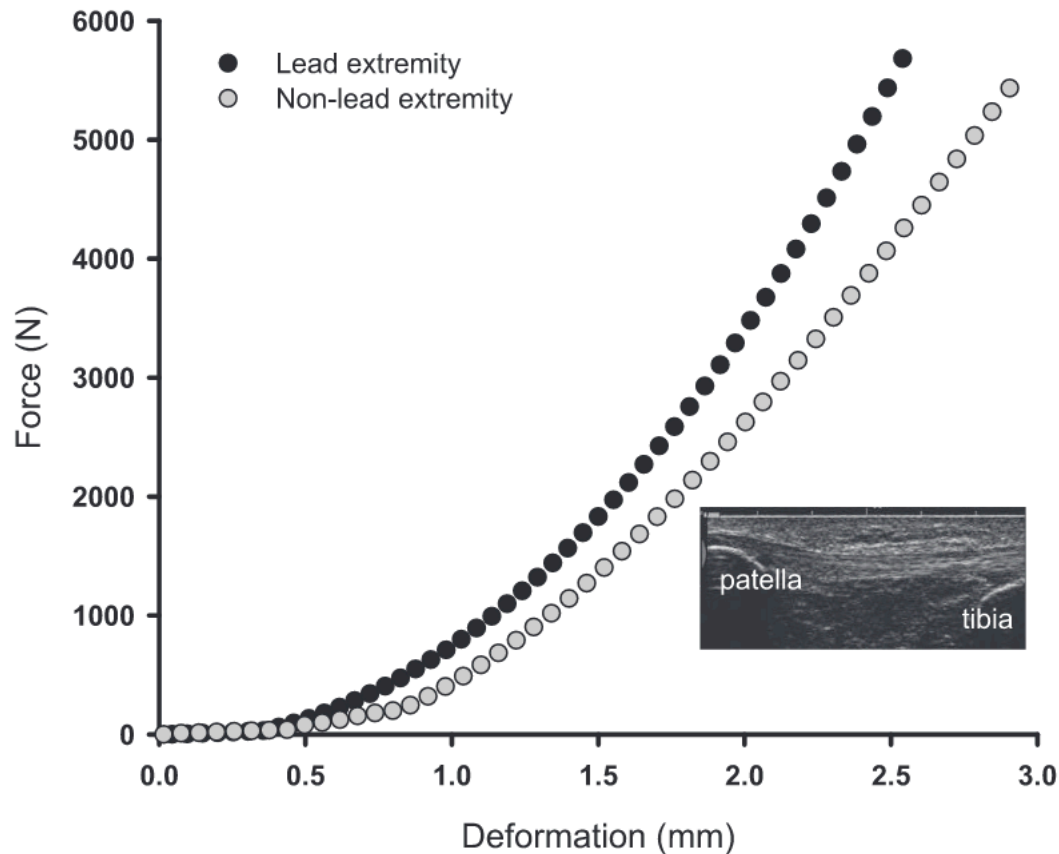


Fig. 3. Patellar tendon force-deformation relationship to a common force.

Context: These two figures were published in a study comparing the properties of the patellar tendon between legs in fencing and badminton athletes. Figure 2 depicts the mean cross-sectional area (CSA), measured proximally, mid-tendon and distally. Figure 3 displays the mean force-elongation relationships.

Figure 2: * means a statistically significant side-to-side difference, # means a significant difference compared to other tendon regions

Questions:

- Examine the figures to describe the differences in tendon properties observed between sides.
- What is the consequence of the side-to-side difference reported in Figure 2 regarding the stress experienced by the tendons during physical activity?
- Explain why a difference is not visible across the entire force-elongation curve in Figure 3.

- Provide an explanation related to the sport these athletes participate in for the different properties reflected in these figures.
- Identify which changes in tendon composition could account for the observed differences.

Answer:

- The Figure 2 shows that the patellar tendon is more hypertrophied in the proximal and distal region in the leading leg. The Figure 3 indicates that the slope of the force-elongation curve is also different in this side, which means a greater mechanical stiffness.
- A larger tendon CSA would limit the stress (force/CSA) experienced by the tendon.
- The force-elongation curves are similar at low force level, in the so-called toe region. This region is not part of the elastic region, which relates to the deformation of the tendon and its stiffness.
- Overall, these differences likely come from a different side-to-side loading pattern for this type of sport, with more loading on the leading leg.
- Tendon hypertrophy comes from an increase in collagen content. The greater stiffness could be linked to this greater collagen content and/or more cross-links.

The kinematics of human running gait

Reference: Willer, J., Allen, S. J., Burden, R. J., & Folland, J. P. (2024). How Humans Run Faster: The Neuromechanical Contributions of Functional Muscle Groups to Running at Different Speeds. *Scandinavian Journal of Medicine & Science in Sports*, 34(8), e14690.

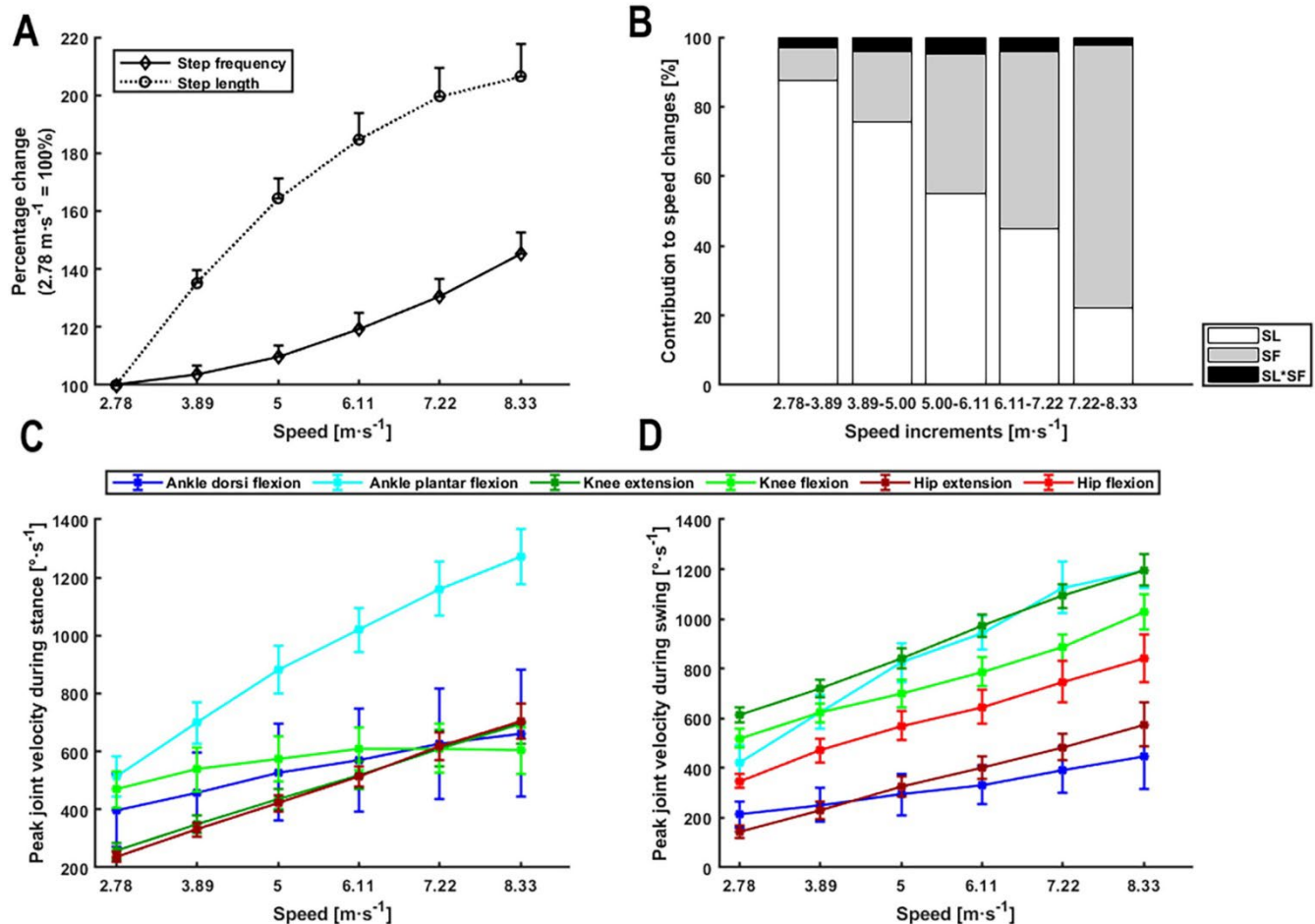


FIGURE 1

(A) Percentage changes in stride frequency and step length normalized to 2.78 m·s⁻¹ at six different running speeds. (B) The contribution of step length, step frequency, and the interaction between both, to each successive increase in speed by 1.11 m·s⁻¹. Peak joint angular velocities of ankle, knee, and hip joints during stance (C) and swing (D) (all shown as positive).

Context: The above figure was published in a study investigating the contributions of step length (SL) and step frequency (SF) to increasing running speeds amongst middle-distance runners. In addition to the spatiotemporal variables (step length and step frequency), the authors wanted to see how joint angular velocities would change to facilitate this strategy.

Questions: Describe what was observed in terms of stride length of stride frequency as speed increased and their relative (%) contribution to the change in speed. In addition, describe knee flexion velocity and ankle flexion velocity and how they were influenced by increasing running speed. If the authors wanted to understand what contributes to a change in angular velocity, which variables might they consider?

Answer guide:

Both step frequency and step length increased non-linearly with increasing speed. The increase in step length leveled off at faster speeds, whereas the increase in step frequency increased at faster speeds. Therefore, the relative contribution of step length to increasing speed decreased at the faster speeds whilst step frequency did the opposite.

Knee flexion velocity increased slightly between the slowest speed and the first increments in speed but plateaued at faster speeds. Ankle plantar flexion velocity increased with increased running speed and the magnitudes were much greater than the knee flexion velocities. To understand what contributes to a change in angular velocity, the authors could look at the total range of motion of the angle as well as the time during which the change occurred to establish whether there was a change in range of motion or a similar range of motion but over a different time interval, or both.